child's HIV status would expedite preventive action by the parent or physician of an immunocompromised HIV-positive student when there is a school epidemic. The cost of even limited disclosure, however, will often be the needless ostracism of the child from school. Information about a child's immunodeficiency, regardless of its cause, should be disclosed to certain care givers, but a child's HIV-seropositive status is to remain confidential information. Exceptions may be to inform those staff members who directly care for children with frequent biting behavior or children incapable of controlling body secretions.

The responsibility of the schools goes beyond safely enrolling HIV-seropositive children in their classrooms. Given that vaccine development is not imminent, community education is the most efficient means of controlling the spread of HIV, and schools have the capability and responsibility to provide this education. Guidelines for an educational curriculum directed towards various grade levels are available from the Centers for Disease Control to educators who want an effective strategy to teach students how to avoid HIV infection.

HOWARD TARAS, MD
San Diego

REFERENCES

American Academy of Pediatrics Committee on Infectious Diseases: Health guidelines for the attendance in day-care and foster care settings of children infected with human immunodeficiency virus. Pediatrics 1987; 79:466-471

American Academy of Pediatrics Committee on School Health and Committee on Infectious Diseases: School attendance of children and adolescents with human T lymphotropic virus III/lymphadenopathy-associated virus infection. Pediatrics 1986; 77:430-432

Guidelines for effective school health education to prevent the spread of AIDS. MMWR 1988; 37(S2):1-14

Rogers MF, Thomas PA, Starcher ET, et al: Acquired immunodeficiency syndrome in children: Report of the Centers for Disease Control national surveillance 1982 to 1985. Pediatrics 1987; 79:1008-1014

Management and Ethical Dilemmas of Diagnosing and Treating Fetal Heart Disease

MAJOR ADVANCES IN ULTRASOUND IMAGING over the past ten years currently provide a systematic approach to evaluating the anatomy, physiology, and rhythm of the fetal heart, with detailed imaging available as early as the 14th to the 15th week of pregnancy. Pediatric cardiologists, perinatologists, and radiologists, often working together, have diagnosed almost every major form of congenital heart disease correctly before birth. Such notable success has not come easily, and, in general, physicians working in this area have often required two to five years of extensive experience to achieve proficiency using ultrasound for a detailed cardiovascular evaluation before birth.

Indications for fetal cardiac ultrasonography include maternal diabetes, maternal drug exposure, maternal infection, a family history of congenital heart disease in a parent or sibling, or persistent fetal arrhythmia. The largest "pick-up" rate for major forms of congenital heart disease will occur if obstetricians evaluate a four-chamber cardiac view as a part of their routine obstetric ultrasonographic screening procedure. They should see the atrial and ventricular septa and four chambers relatively symmetrically dividing the cardiac ultrasound "silhouette" in this plane. Perhaps 90% of the major forms of congenital heart disease can be ruled out by a normal four-chamber view.

Alternatives available in the aftermath of the diagnosis of congenital heart abnormality may include pharmacologic therapy for an arrhythmia; counseling a family about the natural history—catheterizations, heart operations, and the like—of infants with similar forms of congenital heart disease after birth; and counseling related to terminating a pregnancy in the appropriate circumstance or making arrangements for the timing and method of delivery for the appropriate medical or surgical intervention after birth, including consideration of a neonatal heart transplantation for certain inoperable conditions. Major additional capabilities for evaluating the fetal heart have arisen with the application of the Doppler to evaluate cardiac function in cases of valvular disease, and high-resolution color flow mapping has assisted in evaluating septal defects as well as the aortic arch and conal truncal abnormalities.

The field of fetal cardiology is rapidly advancing. It requires the highest level of technical skills and cooperation among obstetric, pediatric, neonatal, ethical, legal, genetic, and surgical colleagues. Several tertiary hospitals have developed centers for fetal diagnosis and treatment where such multidisciplinary expertise can be coordinated, not only for cardiac but for other organ system diseases diagnosed and potentially treated in the fetal stage. Through regionalized team efforts, advances in this dynamic area of fetal medicine will be controlled and yet nurtured to encourage further advances.

DAVID J. SAHN, MD
San Diego

REFERENCES

Allan LD, Crawford DC, Anderson RH, et al: Spectrum of congenital heart disease detected echocardiographically in prenatal life. Br Heart J 1985; 54:523-526

Kleinman CS, Donnerstein RL, Jaffe CC, et al: Fetal echocardiography—A tool for evaluation of in utero cardiac arrhythmias and monitoring of in utero therapy: Analysis of 71 patients. Am J Cardiol 1983; 51:237-243

Reed KL, Sahn DJ: A proposal for referral patterns for fetal cardiac studies. Semin Ultrasound CT MR 1984; 5:249-252

Sahn DJ: Real-time two-dimensional Doppler echocardiographic flow mapping. Circulation 1985; 71:849-853

Sahn DJ, Lange LW, Allen HD, et al: Quantitative real-time cross-sectional echocardiography in the developing normal human fetus and newborn. Circulation 1980; 62:588-597

Silverman N, Kleinman CS, Rudolph AM, et al: Fetal atrioventricular valve insufficiency associated with nonimmune hydrops: A two-dimensional echocardiographic and pulsed Doppler ultrasound study. Circulation 1985; 72:825-832

Exogenous Surfactant for the Respiratory Distress Syndrome

SINCE THE 1970s, therapy for the neonatal respiratory distress syndrome has focused on metabolic resuscitation and assisted ventilation of preterm infants with ventilatory failure until the gene controlling surfactant production and secretion is activated. Surfactant deficiency is central to the pathophysiology of the respiratory distress syndrome. Successful exogenous surfactant administration has led to the development of several promising surfactants for exogenous use.

Human surfactant, derived from amniotic fluid, contains all surfactant proteins (SP-A, -B, and -C), phospholipids identical to "mature" adult surfactant, and a small percentage of neutral lipids. Human surfactant instilled into the airways has been shown to reverse ventilatory failure from the respiratory distress syndrome; to bring about a rapid improvement in oxygenation; and, in very premature infants, to decrease the mortality and the prevalence of bronchopulmonary dysplasia.

Several "first-generation" surfactants have shown promise in clinical trials. Surfactant TA, calf lung surfactant extract, and human surfactant are currently being evaluated in multidose clinical trials to establish efficacy and safety. Synthetic surfactant, not derived from natural sources and

void of surfactant protein (Exosurf), is concurrently being studied in similar trials. Both preventilatory treatment and postventilatory "rescue" trials of surfactant treatments are being systematically investigated using these surfactants. Using human surfactant has thus far been free of complications, and the two-year neurodevelopmental follow-up evaluation suggests an overall improvement in treated infants.

Critical advances in isolating surfactant components and cloning suggest that recombinant surfactants, comprising critical proteins or biologically active peptides and phospholipid mixtures in ideal combinations, will become "second-generation" surfactants.

Until the efforts of genetic engineering are successful, neonatologists will be limited to using natural surfactant derivatives or artificial surfactants in carefully controlled trials sanctioned by the Food and Drug Administration. Of course, only premature infants with evidence of surfactant deficiency should receive this experimental therapy. If the present trials are successful, surfactant therapy should by 1990 become a mainstay of the treatment of the respiratory distress syndrome.

T. ALLEN MERRITT, MD
MIKKO HALLMAN, MD
San Diego

REFERENCES

Edwards DK, Hilton SV, Merritt TA, et al: Respiratory distress syndrome treated with human surfactant: Radiographic findings. Radiology 1985; 157:329-334

Fujiwara T, Chida S, Watabe Y, et al: Artificial surfactant therapy in hyaline-membrane disease. Lancet 1980; 1:55-59

Hallman M, Merritt TA, Jarvenpaa AL, et al: Exogenous human surfactant for treatment of severe respiratory distress syndrome: A randomized prospective clinical trial. J Pediatr 1985; 106:963-969

Merritt TA, Hallman M, Bloom BT, et al: Prophylactic treatment of very premature infants with human surfactant. N Engl J Med 1986; 315:785-790

Revak SD, Merritt TA, Degryse E, et al: Use of human surfactant low molecular weight apoproteins in the reconstitution of surfactant biologic activity. J Clin Invest 1988; 81:826-833

Vaucher YE, Merritt TA, Hallman M, et al: Neurodevelopmental and respiratory follow-up in early childhood following human surfactant treatment. Am J Dis Child, in press

Bronchopulmonary Dysplasia (Infantile Chronic Lung Disease)

WITH THE INCREASED SURVIVAL of neonates who have severe respiratory insufficiency, bronchopulmonary dysplasia has become an important cause of chronic illness during the first five years of life. Causes of respiratory tract injury in early infancy that may lead to bronchopulmonary dysplasia include lung immaturity (hyaline membrane disease); aspiration syndromes; infection, especially viral; and respiratory tract malformations associated with impaired mucociliary function and chronic airways soilage. These infants survive with impaired mucociliary clearance mechanisms, increased bronchial reactivity and bronchomotor tone, and impaired lung water dynamics.

While diuretic and bronchodilator therapy continue to be mainstays of management, recent studies have shown the importance of adequate oxygenation for the recovery from bronchopulmonary dysplasia. Maintaining an arterial oxygen tension above 85 torr is desirable. Occult, sometimes life-threatening hypoxemia has recently been described in patients with bronchopulmonary dysplasia who have relatively mild clinical disease. Such infants benefit from a predischarge evaluation of gas exchange in all activity states by continuous transcutaneous monitoring of blood gas measurements so that supplemental oxygen can be prescribed for vulnerable periods during daily activities.

Lung reinjury must be avoided. Intercurrent infections—including extrapulmonary infections—should be treated aggressively, and influenza prophylaxis and treatment should be undertaken for any infants older than 6 months. Pertussis immunization is essential even for those infants receiving anticonvulsant therapy. Gastroesophageal reflux must be treated aggressively to avoid aspiration reinjury. Environmental sources of lung injury such as pollution, side-stream cigarette smoke, and exposure to talc should be avoided.

The recovery from bronchopulmonary dysplasia is associated with normal growth and repair of the respiratory tract. Growth failure frequently occurs as a consequence of chronic hypoxemia, increased oxygen consumption, and an inadequate intake of protein and other calories compared with the increased caloric expenditure. Therefore, continuing nutritional assessment and management are important.

CHERYL D. LEW, MD
Los Angeles

REFERENCES

Bader D, Ramos AD, Lew CD, et al: Childhood sequelae of infant lung disease: Exercise and pulmonary function abnormalities after bronchopulmonary dysplasia. J Pediatr 1987; 110:693-699

Garg M, Kurzner SI, Bautista DB, et al: Oxygen desaturation during sleep and feeding in bronchopulmonary dysplasia. Clin Res 1987; 35:234A

Kurzner SI, Garg M, Bautista DB, et al: Growth failure in bronchopulmonary dysplasia: Elevated metabolic rates and pulmonary mechanics. J Pediatr 1988; 112:73-80

Kurzner SI, Garg M, Bautista DB, et al: Growth failure in infants with bronchopulmonary dysplasia: Nutrition and elevated resting metabolic expenditure. Pediatrics 1988; 81:379-384

Transient Erythroblastopenia of Childhood

TRANSIENT ERYTHROBLASTOPENIA OF CHILDHOOD (TEC) is characterized by the gradual development of anemia—a hemoglobin level of 2 to 8 grams per dl—reticulocytopenia, and a pronounced reduction of bone marrow erythroblasts. The platelet count is normal to increased. The leukocyte count is usually normal, although 20% of children may have significant neutropenia (less than 1,000 neutrophils per μl). The disorder occurs in previously healthy young children from 6 months to 4 years of age and is seen with equal frequency in boys and girls. Occasionally cases of the disorder occur in clusters, suggesting it may be a consequence of some seasonal environmental toxin or virus. To date, however, serologic studies have failed to reveal exposure to a common virus. Published data suggest that anemia is due to a transient antibody-mediated suppression of normal erythropoiesis.

The natural history of TEC is that all patients recover spontaneously in a few weeks and there are no long-term hematologic sequelae. In many children with the disorder, particularly if there is evidence of recovery at the time of diagnosis, no specific therapy other than careful observation is necessary. Erythrocyte transfusions are indicated only if a child is symptomatic from the anemia. Neither iron nor steroid therapy has any role in the management of this disorder.

The diagnosis often is confused with that of iron deficiency anemia, although the erythrocytes in patients with TEC are normocytic (mean corpuscular volume [MCV], 70 to 85 fl), whereas iron deficiency anemia is characterized by microcytosis (MCV, 50 to 70 fl). Transient erythroblastopenia of childhood also may be confused with Blackfan-Diamond anemia, although the latter generally presents before 6 months of age, often is associated with congenital abnormalities, and invariably is characterized by macrocytic erythrocytes (MCV, 85 to 100 fl) with many fetal-like features. Because some children with TEC also have significant neu-